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Oedema of the Lungs in Drowning

The use of expired air for resuscitating the apparently dead goes back at least to biblical times. When Elisha was faced with a respiratory emergency:

'He went up, and lay upon the child, and put his mouth upon his mouth . . . and the flesh of the child waxed warm.' (II *Kings*, 4, 34).

Mouth-to-nose resuscitation is regarded as even more efficient than the mouth-to-mouth technique and these procedures have to-day been adopted almost universally.

In the use of these methods in cases of drowning, stress is laid upon the need to begin resuscitation even before the victim has been removed from the water and while he is still on his back. This means, in effect, that in the past undue emphasis may have been placed upon the mechanical effects of the presence of water in the respiratory tract.

Cot[1] suggested that death from drowning may be caused as much by fulminating oedema of the lungs as by the aspiration of fluid. In a more recent review Swann[2] stated that, in cases of drowning, the oedema was fairly extensive throughout the lungs. These observations, coupled with the efficacy of modern methods of expired-air artificial respiration, draw attention to the need for a revision of our concepts of the pathology of drowning. Insufficient attention appears to have been paid to the importance of oedema of the lungs.

This oedema is also the probable explanation of the frothy fluid found in the respiratory tract in cases of drowning.

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1. Cot, C. (1931): *Les Asphyxies Accidentelles*, N. Maloine, Ed., Paris. *Quoted by* Swann, H. G. and Spafford, N. R. (1951): Texas Rep. Biol. Med., **9**, 356.
2. Swann, H. E. (1960): A.M.A. Arch. Path., **69**, 557.

Acute pulmonary oedema can, of course, be due to many other causes. Its evaluation may therefore present great difficulty, particularly if the circumstantial evidence of drowning is not clear or available. It is necessary to distinguish death occurring in association with submersion from death actually due to drowning. Moritz[3] has put the position very fairly:

'To conclude that death has been due to drowning simply because the body was found in the water is likely to result in civil injustice or in the non-recognition of crime.'

3. Moritz, A. R. (1954): *The Pathology of Trauma*, 2nd ed., p. 190. Philadelphia: Lea and Febiger.

Faecal Matter in Stains

Their Identification*

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Owing to the frequent finding of faeces at the scene of a crime Moeller[12] in 1897 suggested that the first specimen of faeces obtained from a person arrested for a crime should be stored for possible examination later. He believed that this in many cases would shorten the criminal investigation. Two years later M. L. Q. van Ledden-Hulsebosch[7] published *Makro- und mikroskopische Diagnostik der menschlichen Exkremente,* in which he described the characteristics of faeces in order to facilitate the identification of specimens. The anatomy of the most common foodstuffs was reviewed, and special emphasis was placed on the microscopic morphology of the remains of the foodstuffs in the faeces, particularly of vegetables. The book is very instructive, and contains 255 photomicrographs. These are, however, old-fashioned, and not of a satisfactory standard from a modern point of view. C. J. van Ledden-Hulsebosch[6] constructed a coprolysator, with the aim of separating particles of faeces according to their size.[6, 14]

There are two aspects to the problem of examination of faeces. The first is to prove that a specimen really is faeces, and the second is to identify the particular specimen. The problem may be easy to solve with large quantities of faeces, but if only small stains are available there may be several difficulties.

It is a curious fact that the examination of such a common human product as faeces is not mentioned in the common textbooks of forensic medicine although such an examination has, in some cases, played an important role in criminal investigations.[1, 2, 4-6, 8, 9, 13] These cases will be reviewed shortly in the discussion of the present case.

Case Report

A 14-year-old girl was found raped and murdered (by strangulation). She had been a virgin, and the defloration had caused some bleeding, so that her private parts were soiled with blood. Around the anus faeces were abundant. She was partly undressed, and her knickers were lying beside

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* Read in an abridged form before the Second International Meeting of Forensic Pathology and Medicine, 21 September 1960, New York, N.Y.

† Head: E. Waaler, M.D.

the body, covered with faeces. Clearly the girl had defaecated before she was undressed. Her vest was also lying beside the body, crumpled and with several large stains of faeces as if it had been used as a towel. It seemed probable that the culprit had been soiled with faeces.

Fourteen days after the girl was found (17 days after the murder) the girl's and a suspected man's clothes were received for examination. On the clothes the police had marked numerous stains for examination for seminal fluid, blood and faeces. Only the examination for faeces will be reported.

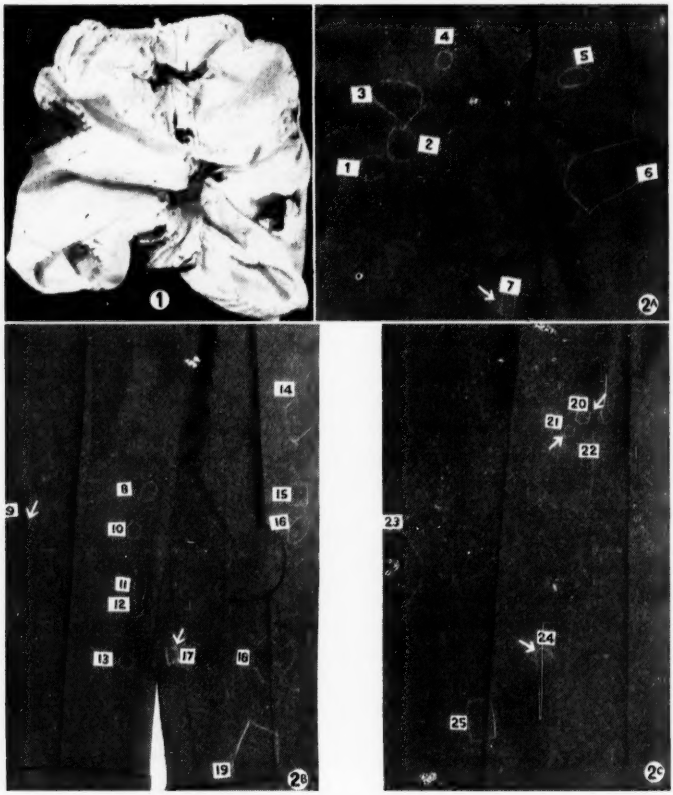

Fig. 1. The girl's knickers with stains and lumps of faeces.
Fig 2. The suspect's trousers.
A: Upper front.
B: Lower front.
C: Lower back.
Stains Nos. 7, 9, 17, 20, 21 and 24 looked like faeces.

The girl's faeces were the usual brown colour (Fig. 1). They contained some nonspecific black granules and several yellowish fragments of fibres of different size. The consistency was solid, and a little brittle because of drying. For the same reason there was hardly any odour. Both the benzidine reaction for blood and the sublimate reaction for bile pigment were negative. A lump of faeces was mixed with water and examined microscopically by the usual clinical methods. In unstained preparations some vegetable matter and bacteria were found, but no convincing muscle fibres and no pathological constituents. In stained preparations a few iodophilic granules (starch) and a few sudanophilic granules (fat) were found, but no iodophilic bacteria. Thus the girl's faeces were completely normal. The negative reaction for bile pigment in spite of the brown colour of the faeces must be ascribed to the fact that there had been exposure to air and light for 2½ weeks before examination. Several tests were performed and all were negative. The method was tried on fresh faeces from a corpse and in this case the reaction was positive.

All the stains on the suspect's clothes were examined with a stereomicroscope at a magnification up to 40 times, in which way a very good view of the stains was obtained. Several stains looked like faeces. (Figs. 2 and 3). There were 6 stains on his Terylene trousers; one stain on the inside of the front of his vest, in the centre of the lowest part; two stains on the fly of the underpants and one on the right shoe. Most of these stains were very small, only a few millimetres in diameter, and as the trousers were a blue-black colour, the stains on these were hardly visible to the naked eye. On the underpants the stains were a little larger. All the stains were completely odourless, and of a similar appearance. They consisted of small brown-yellow flakes, dry and a little glossy with a few black granules and yellow fragments of fibres. In appearance they were identical to smears of the girl's faeces that we made on the suspect's clothes. The benzidine reaction and sublimate reaction were negative in scrapings of the stains.

Neither the girl's faeces nor the scrapings of the stains gave any fluorescence in ultra-violet light. Both showed a few doubly-refracting granules in polarized light.

Examination for Vegetable Matter. A lump of the girl's faeces was mixed with 10% potassium hydroxide and placed in an incubator at 50° C. for 24 hours. By this procedure all organic material is digested, except vegetable tissues. Several unstained preparations were made from the sediment, and in these a series of vegetable particles were recognized. If one wants to preserve the preparations, they must not be covered with Canada balsam, as the KOH produces a white precipitate that obscures the preparation. Glycerine may be used. A botanist was consulted (Professor

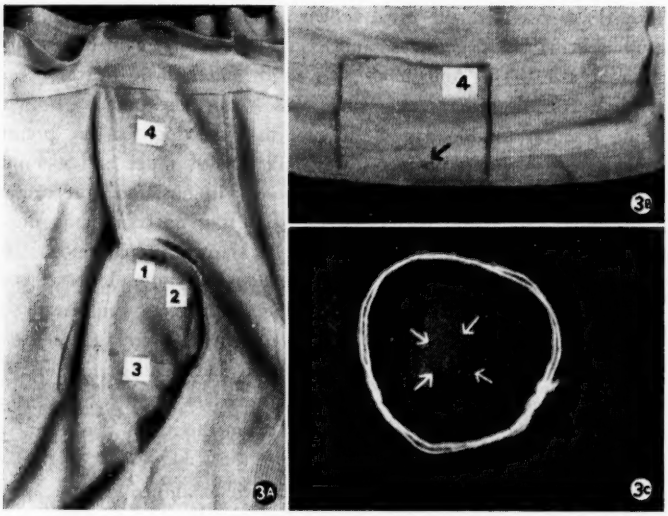

Fig. 3. The suspect's other clothes.
A: Front of the underpants.
B: Centre front, inside, lowest part of the vest.
C: Right shoe.
Stains Nos. 1 and 2 on the underpants, stain No. 4 on the vest and the stain on the shoe looked like faeces.

Knut Faegri, University of Bergen). He classified the particles as follows (Fig. 4, A-D):

1. Stone cells, partly isolated, partly combined into units of 2 or more cells.
2. Fragments of vascular bundles, that may be found in the peripheral nerve-endings of leaves. Spiral thickenings were seen isolated from the vascular bundles.
3. Fragments of cortical-like cells probably from a root.
4. Remains of cell units.

The botanist pointed out that the vegetable matter appeared to have gone through a putrefactive or digestive process.

Of these vegetable remains only the stone cells are, to some extent, specific, as they are not a common finding in vegetables. They may be found in pears, dates, pepper, pimento and cherry stones. The other remains are highly nonspecific, as they may be found in several different vegetables. The botanist, however, expressed the opinion that, even if the vegetable remains were not specific, the finding of the same mixture of vegetable remains in 2 specimens of faeces strongly suggested that these 2 specimens were identical.

Preparations of the scrapings of the stains on the suspect's clothes were made in a similar manner to the preparations of the girl's faeces. In these preparations all the above-mentioned elements were found (Fig. 5), and

there were no elements in the stains that could not be found in the girl's faeces.

The back of the suspect's underpants was soiled on the inside with faeces, undoubtedly his own. Several preparations were made from these stains. Primitive vascular bundles and remains of cell units were found, but no stone cells or cortical cells.

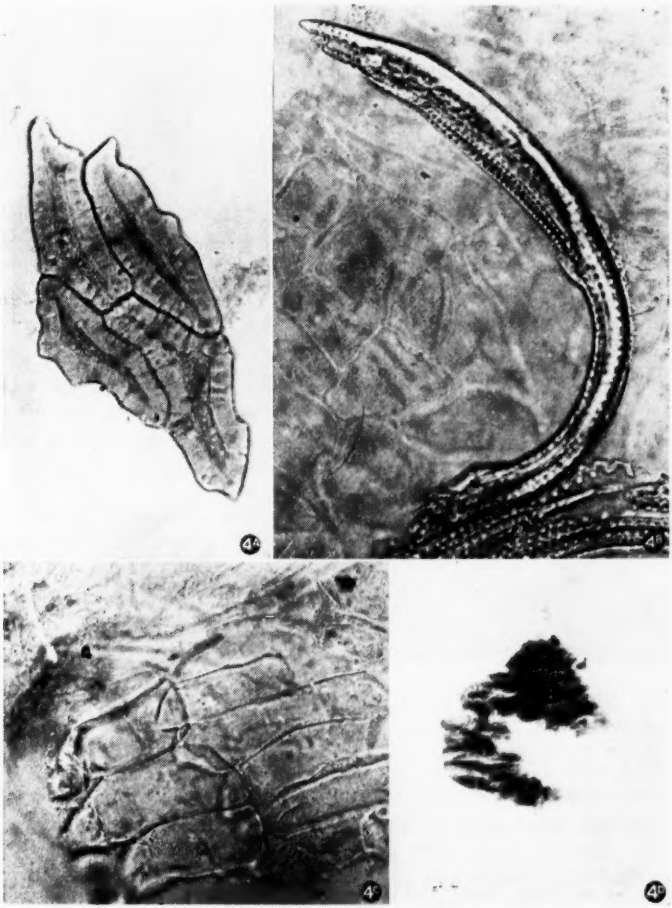

Fig. 4. Vegetable tissue from the girl's faeces.
A: Stone cells.
B: Vascular bundles.
C: Remains of cell units.
D: Cortical cells (x 350).

Bacteriological Examination. Due to the other examinations it was not possible to examine all the stains bacteriologically. Therefore 2 stains were chosen at random (one from the trousers (stain No. 21) and one from the underpants (stain No. 2), cut out with scissors and, together with

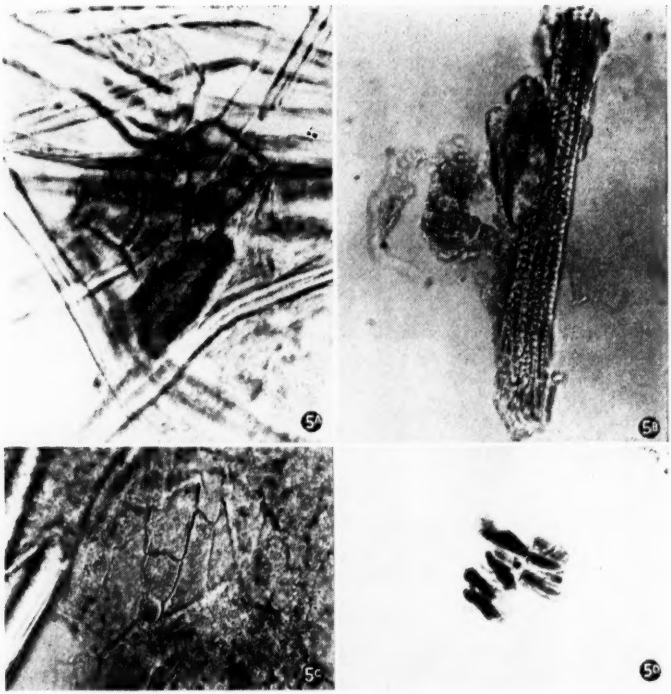

Fig 5. Vegetable tissue from stains on the suspect's clothes.

A: Stone cells.
B: Vascular bundles.
C: Remains of cell units.
D: Cortical cells (x 350).

(The large fibres in A and C are from the fabric of the underpants.)

a lump of the girl's faeces and a part of the suspect's underpants with his own faeces, were examined bacteriologically (Professor Per Oeding, the Gade Institute, University of Bergen). The problem was: Could faecal bacterial flora be found in the stains, and could these be identified with the flora in the girl's faeces or the suspect's faeces?

Results:
The girl's faeces:
E. coli;
Enterococci, 2 types;
Staphylococcus albus, coagulase negative;
B. subtilis.
The suspect's faeces (from his underpants):
Enterococci, 2 types;
Staphylococcus aureus, coagulase negative;
Sarcinae.
Stain from suspect's trousers:
Enterococci;
Staphylococcus aureus, coagulase positive;
B. subtilis.
Stain from suspect's underpants;
Enterococci;
Staphylococcus aureus, coagulase negative;
B. subtilis.

Enterococci were demonstrated in all 4 specimens, and *B. subtilis* in the faeces from the girl and in both the stains. These bacteria were examined more closely by cultural and biochemical methods. The results did not, however, help to identify the stains, and it was felt that a serological examination was not indicated.

The bacteriologist concluded that the absence of *E. coli* in the examined stains could possibly be due to the fact that the stains were old. The finding of enterococci in the stains indicated the presence of faecal matter.

Discussion

The normal human adult excretes 100–150 g. of faeces in 24 hours. These have a doughy consistency, and a characteristic odour and colour. They are made up of digestive residual products, undigested or partly digested foodstuffs, bacteria, secretions from the glands in the gastro-intestinal tract, various cells (leucocytes and intestinal epithelium) and water. The water content amounts to about 75% of the total weight. About one-tenth of the dry weight is made up of bacteria. All these constituents may vary greatly under both physiological and pathological conditions. It is beyond the scope of this report to discuss these changes. They will be found described in detail in textbooks on physiology and medicine. It should, however, be mentioned that faeces under pathological conditions may contain helminths and protozoa, a fact that may have medico-legal interest.

The means by which one may answer the two previous questions on a medico-legal examination of faeces overlap each other and will be discussed together. The following constituents of faeces are highly nonspecific and are therefore usually not suitable for the purpose of identification:

Digestive residual products; secretions from the glands in the intestinal tract and the various cells that may be found.

It must be admitted, however, that under pathological conditions these constituents may play a role. As the faeces in question were completely normal from a clinical point of view, there remains to be considered:

1. The odour.
2. The colour.
3. Undigested or partly digested foodstuffs.
4. The content of bacteria.
5. Possible pathologic constituents.

1. The odour of the faeces is caused by bacterial activity in the intestines. Although it may vary considerably, it is usually quite characteristic; but in dried specimens, particularly in small stains, it may disappear completely as a result of drying.

2. Normally the brown colour of the faeces depends mainly on the amount of bile pigment and its derivatives (bile pigment converted by bacteriological action), but partly on the quality of the food. Pathological constituents, e.g. blood and certain drugs, may give a pathological colour. The bile pigment and its derivatives may be demonstrated qualitatively by Schmidt's sublimate reaction.*

The demonstration of bilirubin alone is not conclusive, as it may be found in vomit. The demonstration of urobilin, however, must be regarded as proof of the presence of faecal matter.

Asada and Kominami[3] have published a method of performing Schmidt's reaction directly on small stains. Their method was tried in our case, but with a negative result, and Reuter[14] has not obtained satisfactory results with their method.

In our case the reaction was negative both in the girl's faeces and in the stains. This is supposedly due to the fact that faeces had been exposed to light and air for 17 days, whereby a chemical change may have occurred in the bile pigments. As mentioned, the method and the reagent were tried on faeces from a fresh corpse with a positive result.

The colour of the stains was found to be completely similar to the colour of stains that resulted when the girl's faeces were smeared on the suspect's clothes. With a negative Schmidt's reaction, however, this similarity is only suggestive of the presence of faecal matter, as a brown sauce, very commonly used in Norway, gave stains of a similar colour.

3. Both the nature and the amount of undigested or partly digested foodstuffs that may be found in the faeces may naturally vary considerably. Remains of both meat and fish may be found,[7] but it is the vegetable remains that have gathered most interest, as they may be most specific.

The cell walls of vegetable tissues are made of cellulose, and are consequently indigestible. However, the connecting middle lamella (consisting

* Equal parts of faeces and a colourless 5% sublimate solution are mixed in a white cup. Within a few hours bilirubin (unchanged bile pigment) gives a green colour, whereas urobilin (derivative of bilirubin) gives a red colour.

of pectines) is partly digestible, and the remains of vegetables and fruit will thus be influenced by the digestive process. A certain structure may therefore appear in the faeces as more or less isolated cells or cell complexes. The microscopic morphology of the individual cells is generally preserved, and faeces have several times been identified through their vegetable content. C. J. van Ledden-Hulsebosch has reported two cases.[6] Both cases concerned faeces found at the scene of a robbery. In the one, remains of onions and potatoes, in the second remains of bread and liver sausage, were found. In both cases the examination of the faeces helped the police to find the culprit. Gross[2] refers to the case of a murdered girl in whose faeces the remains of figs were found. Fig trees grew only in one garden in the neighbourhood, and in the house belonging to this garden the murderer was located. Johnson[9] reports a case in which the analysis of faecal stains on the shoe of a thief helped to identify him. Faeces at the scene of the crime and the faecal stains on the shoe contained 'vegetable tissue, consistent with the leaves of lettuce, cabbage or other leafy vegetables, and particles of fruit hull or skin.' Hepner[4] has reported two cases in which the finding of burnt crusts, blueberries and onionlike tissue in the first, and burnt crusts and caraway seeds in the second case helped to identify the faeces. In a case of rape and murder reported by Dorenfelt[1] palisade cells from peas or beans were found in the murdered girl's faeces. Her mother informed the police that the girl had eaten pea soup. On the suspect's shoes were found stains that contained palisade cells.

In our case several nonspecific vegetable tissues were found. The stone cells were, however, quite characteristic and easily identifiable by anyone experienced in microscopy, even if he is not a botanist. Special emphasis must be placed on the absence of stone cells (and of cortical cells) in the stains of the suspect's own faeces on his underpants. This practically excludes him as a source of the stains on his other clothes. As the botanist consulted pointed out, emphasis must also be placed on the entire mixture of vegetable remains when comparing 2 specimens of faeces, even if the components are not specific alone.

When our investigation started, the police, at our request, secured information about what the girl and the suspect had eaten during the 3 to 4 days before the murder. This information, however, gave no supportive evidence in our case, as information about the eating of pears or any other fruit that contains stone cells did not come forth. On the other hand, the eating of a pear is not such an unusual incident that it would be mentioned to anyone.

Owing to the fact that all plants contain chlorophyll, Kraft[11] published a method for the demonstration of chlorophyll in faeces. Such a demonstration, however, can hardly have any practical interest as it cannot serve to identify the faeces. A microscopic examination as outlined above is far better.

4. About one-tenth of the dry weight of the faeces is made up of bacteria, mainly *E. coli* and enterococci, and the demonstration of either of these bacteria must be regarded as strong evidence of faecal contamination. Under pathological conditions a vast number of other bacteria may be found.

E. coli may be typed serologically. In Hoen's case[5] a woman and a child were murdered and the woman had defaecated. On the suspect's shoe a stain looking like faeces was found. *E. coli* were found both in the woman's faeces and in the stain. Immune sera were made to these 2 strains and both sera cross-reacted with the strains. It was thus highly probable that both strains originated from the same source. With modern methods the typing of *E. coli* can be done with more certainty and it has been found that one individual's faeces may contain several types of *E. coli*.[9] It therefore seems that if the same types of *E. coli* are found in 2 specimens of faeces, this suggests very strongly that the 2 specimens are identical. A full medico-legal examination of faeces should therefore include a bacteriological examination.

In our case *E. coli* and enterococci were found in the girl's faeces. In public health work the demonstration of *E. coli* in, e.g. drinking water, indicates human or animal faecal contamination. *E. coli* was, however, not found in the stains on the suspect's clothes, not even in undoubted stains of faeces (the suspect's own faeces from the back of his underpants). As the bacteriologist pointed out, this may have been due to the fact that the stains were old. In addition, the suspect's trousers may have been washed (the murder took place on a Saturday evening and the accused admitted that his shirt had been washed the following Sunday morning). The demonstration of enterococci, however, strongly indicates faecal contamination, although these bacteria sometimes may be found in non-faecal material, e.g. sputum. It is to be noted that *B. subtilis* was found in the girl's faeces and in both the stains, but not in the suspect's faeces. *B. subtilis* is, however, so commonly found in dust, etc. that this finding can hardly serve any identification purpose. The finding of staphylococci and sarcinae is non-conclusive. Thus in our case the bacteriological examination gave evidence of the faecal nature of the stains, but it could not be of any help in the further identification of the stains. Therefore, when the police secure faeces or suspected stains of faeces, the medical examiner should be summoned immediately to prevent contamination and to make the necessary arrangements for bacteriological examination.

5. Pathological bacterial content will become evident on examination. In addition, particularly helminths and protozoa may be of interest. Thus, in faeces left at the scene of a murder round worms were found. Among 6 suspected men one had round worms in his faeces.[13] In another case two types of tropical protozoa were found in the faeces left at the scene of a

crime, and the culprit was identified as a pensioned Indian soldier who carried the same protozoa.[6] In a recent case the finding of eggs of round worms and whip worms in the faeces identified the culprit.[8] Various methods may be used to demonstrate helminths and protozoa in the faeces. Vanni[15] has discussed from a legal point of view the characteristics of some of these helminths and protozoa and the methods for their demonstration.

In our case no pathological constituents were found in the faeces.

As a result of our investigation we concluded that the stains examined on the suspect's clothes were proved to be from faeces and that there was ample reason to believe that these stains originated from the girl's faeces.

Summary and Conclusion

A rape and murder case is reported, in which the murdered girl had defaecated. On the suspected man's clothes were found small stains very like stains of faeces. By microscopic examination of the vegetable remains and bacteriological examination these stains were proved to be due to faeces originating most likely from the girl's faeces.

The way in which faeces and stains of faeces may be identified are discussed, and the literature on the subject is reviewed.

It is stressed that a complete medico-legal examination of faecal matter for identification purposes includes:

1. Examination of the odour.
2. Examination of the colour (demonstration of bile pigments and their derivatives, pathological colour caused by blood, drugs etc.).
3. Examination of the content of undigested foodstuffs, particularly vegetables, and classification of these tissues.
4. Bacteriological examination, with typing of the bacteria.
5. Examination for pathological constituents, especially helminths and protozoa.

It is further stressed that when the police secure faecal matter for examination, the medical examiner must be summoned at once to make the arrangements necessary for a complete examination.

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Reviews of Books

Miscarriages of Justice

Miscarriages of Justice. By C. G. L. Du Cann. (1960. Pp. 272 + Index. R 2.10).
London: Frederick Muller Limited.

The author writes from Gray's Inn, London. He has been irritated by the 'botching and patching; the mending and making-do; the perpetual tinkering and tampering which masquerade as genuine reform in criminal law and procedure (of which, parts of the Homicide Act 1957 furnish a melancholy example). These are no real remedy for admitted evils. These make confusion worse confounded. These often create greater evils than they cure. But these are all that our inefficient, oligarchic Governments with their servile, shadow-boxing, tied-houses of Parliament spasmodically provide . . .

What is needed is a permanent Commission in continuous session; doing the great work that Lord Chancellors of genius like Bacon and Westbury wanted done; surveying the whole field; and making recommendations to Parliament. A consolidated and simplified Criminal Code should be one aim. The streamlining and modernization of the criminal courts and their procedures should be another.'

The tone which this crusader for reform has adopted is strident and mars the presentation of his case which, however, he unrepentantly offers as 'a sacrilegious and blasphemous brawler in that holy of holies, the Temple of Justice. It questions, *inter alia,* the alloted *rôles* of the advocates, the judge, and the jury; the accusatory, instead of an inquisitory, procedure; the utility of the dock and the oath in modern times; the efficacy of the single-judge system: in short, the whole set-up. To date, such ancientry has been accepted as though ordained by the Deity as a sacrement and so exempt from all criticism.' Certainly such incidents as the trial of Jessie M'Lachlan in Glasgow in 1862 cause Christina Brand to write in *Heaven Knows Who: The Trial of Jessie M'Lachlan* (p. 121):

'As it was, it was dealt with by a single, hostile judge. British justice blushes for the conduct both before and during the trial of the Jessie M'Lachlan case.'

Mr. Du Cann has addressed himself to the general public in an endeavour to win support for the overthrow of the 'idols of the tribe, the cave, the marketplace and the theatre.'

Despite its irritating prose style, the volume merits serious attention by legal practitioners as well as the citizens who must endure justice as it is meted out in the United Kingdom.

Stewart and Stolman's *Toxicology*

Toxicology: Mechanisms and Analytical Methods. Ed. by C. P. Stewart and A. Stolman. (1961. Volume II. Pp. 851 + Index. With Illustrations. $25.00).
New York: Academic Press Inc., Publishers.

This is the second volume in the encyclopaedic review of toxicology edited by Stewart and Stolman.

The need for such a comprehensive survey has been dictated by the rapid and large increase in the number of potentially toxic substances developed to meet man's needs in industry, agriculture and medicine.

The importance of enzymes in the mode of action of many poisons is recognized by the devotion to this aspect of the matter of a special chapter.

Professor Harger, well-known for his work on the analysis of alcohol in breath samples, contributes an excellent chapter on *Ethyl Alcohol.* This chapter is particularly valuable for the review of the interpretations of the level of the blood alcohol applied in the Scandinavian countries, Switzerland and East Germany, Canada and the U.S.A.

Curry reviews the *Barbiturates,* which account for almost 10% of all drug prescriptions in the United Kingdom. They comprise therefore one of the most common sources of poisoning. Copeman's work on *Poisoning by Barbiturates* (which first appeared in the *Journal of Forensic Medicine* in 1954) is not reviewed. His paper includes valuable data on recovery of barbiturates from viscera removed at autopsy in cases of suicide and overdosage.

Inter alia, pesticides in food, poisonous plants and radio-active isotopes are also surveyed.

Stolman reviews the determination of haemoglobin derivatives, halides, pophyrins and glucuronic acid.

The concluding chapter is on *The*

Treatment of Acute Poisoning, a chapter which gives the volume as a whole a wide appeal.

The massive accomplishment represented by these 2 volumes is a valuable contribution to the literature of analytical chemistry and forensic toxicology. It will undoubtedly be an essential work of reference not only for the laboratory worker carrying out an analysis but also for the expert who seeks to interpret and apply the results obtained.

Abstract

Intra-Abdominal Haemorrhage after Electroshock Therapy

A case is reported of massive intra-abdominal haemorrhage after electroshock treatment under thiopental-succinylcholine anaesthesia.

The use of thiopental-succinylcholine anaesthesia in electroshock therapy has broadened its indications among patients previously considered major risks.

The occurrence of acute circulatory collapse after electroshock therapy should alert neuropsychiatrists to the possibility of an acute surgical emergency, the prompt recognition of which may be life-saving.

[Mancao, M. Pa., Gordon, E. T. and Goldman, Max (1960): New Eng. J. Med., **264**, 609].

British Association in Forensic Medicine

Proceedings of the Winter Meeting held at Kings College Hospital, Denmark Hill, London, S.E.5, on 21 November 1959

Symposium: The Association of Trauma and Disease

Introduction: Dr. F. E. Camps

The association of trauma and disease has always been and always will be a problem, perhaps even a greater problem in the U.S.A. than it is here owing to the different approach to the question of compensation. Following the usual policy of the Association we are not taking this question from the point of view of every-day material and our Secretary has collected together people who are experts on their various subjects. I think it best to take all the papers and then have a discussion at the end. In this way we can eliminate the possibility of one paper having to be cut short because of lack of time.

Some Remarks on Chemical Injury of the Liver

Its Relationship to Disease

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At least 5 forms of liver necrosis can now be recognized, although all have many points in common and, indeed, the underlying mechanisms may be shared by each.

1. *Hyperacute necrosis* may set in at once after trauma or chemical intoxication.

My interest in this was aroused by finding, some years ago, massive white infarction of the liver in a young man who died 24 hours after a severe alcoholic bout. Autopsy disclosed a large gastric ulcer, but no obstruction or disease of the hepatic vessels and I wondered whether there had been absorption of much alcohol from the ulcer bed and its carriage to the liver in quite large quantities or rapidly, by the portal vein. When my colleagues and I injected alcohol or a variety of other organic compounds like $CHCl_3$ or CCl_4 directly into the portal vein of rabbits white infarction was produced almost instantaneously without portal vein thrombosis.

2. *Acute necrosis* may develop speedily over hours, especially when certain organic solvents are inhaled in high concentrations or absorbed by the stomach and maybe through the skin. Chloroform and carbon tetrachloride are classical examples. DDT and tannic acid are effective in large doses. Necrosis from these can be detected in 6–12 hours grossly and long before this microscopically. It is hurried up or accentuated by malnutrition, fatigue and haemorrhage. Prolonged childbirth sometimes favours speedy liver necrosis; thyrotoxicosis occasionally is a factor. I have wondered whether emotional upset contributes through circulatory disturbance in the liver. Vaso-dilatation of hepatic vessels, known to occur then, would allow a poison an opportunity to act on the liver cells because of slower flow past these cells.

3. *Slow-acting necrosis* is the outcome most often of repeated exposure to poisons. TNT may give extensive liver damage with much regeneration in 4–6 weeks; some of the organic solvents take months or even years to exert a cumulative action. Here the possibility of associated viral or even bacterial infection has to be considered and these may accelerate the onset of one or other forms of cirrhosis.

4. *Delayed necrosis* is best known from animal studies. These first of all suggested that diets deficient in protein or S-amino acids and vitamin

B_{12} could precipitate liver necrosis after some weeks. It is now known that the key to the necrosis is deficiency of vitamin E (α-tocopherol) and that a low protein diet assists. Selenium salts can protect against necrosis. Selenium has turned out to be the responsible agent when S-amino acids are used to protect animals on poor protein diet, for it is invariably present as a contaminant of these amino acids.

I suspect that this form of necrosis does develop from time to time in man in parts of the world where under-nutrition is common, but more often fatty degeneration of the liver and cirrhosis are the outcome. It takes weeks or months to develop but recently Dr. A. Maclean in my Department has obtained evidence that chemical changes develop in liver cells long before necrosis is apparent.

5. *Viral necrosis* may be hyperacute and massive with the picture of acute yellow necrosis, or may smoulder along with many mild or moderate outbreaks over weeks or months to give a mixed picture of destruction and repair. It is stirred up by alcohol or another acute infection or fatigue. I also suspect that hormonal upsets may predispose a subject to viral outbreak.

In most of these cases the exact mechanism has not yet been worked out, but with chloroform and carbon tetrachloride we now know that they give bottle-necks at various stages in the liver cell metabolism of fats and carbohydrates and that the cell may pass into a condition of relative choline insufficiency which leads to fatty degeneration, because the poison stimulates overproduction of phosphatidic acid in the cell (unpublished work of Dawkins, Judah and Rees). I must also emphasize the importance of latent infections, especially in young people, and the contributory part of fatigue. The former is more apparent in acute cardiac and pulmonary disease than with the liver. I have seen sudden pulmonary oedema in young soldiers collapsing during heavy duties in which unsuspected mild rheumatic infection of the heart was discovered at autopsy. I have also encountered a few cases of unexpected patchy liver necrosis after thyroid operation in which the liver showed mild, though distinct, chronic hepatitis. I have wondered, too, whether the allergic state induced by some drugs makes the liver susceptible to necrogenic agents. In a number of cases of chlorpromazine jaundice functional hepatic disturbances were far out of proportion to the structural damage in the liver.

Forensic pathologists have a wonderful opportunity of helping us to disentangle this mixed group of liver and other organ injury if only by recording accurately the presence of pre-existing disease or contributory factors such as mental disturbance, allergy, fatigue, dietary errors and the like.

The Relationship of Trauma to Tumour Formation

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Trauma, in the sense of a single mechanical injury, has often been blamed for the subsequent development of a tumour. Unfortunately the proper scientific evaluation of any such cause-effect relationship has been confused by the publication of numerous clinical anecdotes, the majority being single case reports in which a tumour is naively assumed to have been caused by an antecedent injury. Examples are: gastric and colonic cancers following abdominal crushing; tumours of the kidney after blows on the flank, plasmacytoma following bone injury and carcinoma of the bronchus after blows on the chest. There are also a number of cases in which soft-tissue sarcomas have been attributed to antecedent injury but, when one weighs the tremendous number of such injuries against the comparative rarity of these neoplasms, it is apparent that the aetiological significance of the trauma is doubtful in most cases. The possibility of trauma simply drawing attention to a growth already present is, of course, widely appreciated. As Ewing put it: 'Traumas reveal more malignant tumours than they cause'.

Should we then dismiss trauma altogether as a factor in the production of new growths? I do not think so; but in order to throw some light on this very controversial subject, it is necessary to extend our concept of trauma to include various types of chronic tissue injury resulting from such agencies as micro-organisms, thermal burns, chemical burns and vascular stasis. For example, the scarring of syphilitic glossitis has long been recognized as an important factor in the pathogenesis of carcinoma of the tongue. With the falling incidence of tertiary syphilis, cancer affecting the anterior two thirds of the tongue in men has become less frequent, whilst a relative increase has occurred among women in whom the neoplasm tends to develop in the posterior third. Again, squamous cell carcinoma sometimes develops at the edge of a chronic varicose ulcer. Stout has reported 36 patients in whom fibrosarcomas originated in scar tissue or at the site of previous infection. Let us consider a few other examples and see what common features emerge.

1. *Thermal Burns.* The development of tumours in burn scars is well authenticated. The neoplasms are usually squamous cell carcinomas of epidermal origin, although fibrosarcomas have occurred in a few cases. Treves and Pack found the average duration of the scar, before the appearance of the cancer, to be 32.5 years. However, in elderly people, whose skins were already hyperkeratotic, this 'incubation period' was greatly reduced and the carcinomas often appeared within quite a short time.

2. *Lupus vulgaris.* Again most of the tumours complicating this condition are squamous cell carcinomas. It is of great interest that the 'incubation period' is shortened in those patients treated by irradiation. For example, Lacassagne found it to be 38 ± 5 years in those who had received no irradiation, but 25 ± 2.7 years in the irradiated patients.

3. *Osteomyelitis Sinuses.* Bereston and Nye have reviewed the cases in which tumours have complicated chronic osteomyelitis. The sinuses have usually been present 20, 30 or more years before the cancer appears. The tibia has been the most common site. In one very interesting case, the lesions were bilateral and had followed compound fractures many years before. The usual type of neoplasm is a squamous cell carcinoma, but in one case at the Royal Marsden Hospital, a fibrosarcoma had developed.

4. *Strictures of the Oesophagus.* The great majority of oesophageal cancers develop in the absence of any known predisposing anatomical abnormality. However, Jackson estimated that some 5% of patients gave a history of long-standing dysphagia before the onset of malignant obstruction and more recently Steiner has stated that about 5% of carcinomas follow organic lesions such as strictures, hiatus hernia or diverticulum. The strictures caused by lye burns are particularly interesting. In many instances the lye (a white opalescent fluid) has been swallowed by young children who mistook it for milk. Lye has also been used by bootleggers to give a pungent taste or 'bite' to diluted liquor. Bigger and Vinson observed 7 cases of cancer among 200 patients suffering from lye strictures, whilst Kiviranta believes that one third may develop carcinoma if they live 30 years or more after the original burning. The following points are noteworthy:

(*a*) The carcinoma usually develops at the site of the stricture;

(*b*) The neoplasm is always a squamous cell carcinoma;

(*c*) There is a long latent period of 16–42 years;

(*d*) The average age of the lye cancer patients is considerably below that of oesophageal cancer cases as a whole; and

(*e*) The predominance of men over women is not so great as it is in the general group of all oesophageal cancers.

There is one other type of chronic tissue injury that I would like to mention: chronic lymphatic stasis. Some years ago Stewart and Treves described a number of cases of lymphoangiosarcoma arising in the oedematous arms of patients who had undergone radical mastectomy. The tumours appear to arise from the hypertrophied lymph vessels and they are often multicentric, developing as widely-scattered haemorrhagic foci in the skin and subcutis. They generally appear a long time (a decade or more) after the mastectomy. These tumours are of interest here because they indicate another way in which trauma may be related to neoplasia: through the intermediary of chronic lymphoedema.

We must always remember that even when the relationship between trauma and neoplasia appears to be definitely established, other factors may be at work. Take, for example, the question of trauma and malignant melanomata. Many people believe that there is an aetiological link between the two and I would certainly not deny that there may well be a connexion. However, the question is by no means simple. Consider melanomata of the foot. Most of these occur on the sole and this fact might immediately suggest the importance of trauma. But, whereas most of the body weight is borne by the heels and metatarsal heads, melanomas do not usually occur in these situations: they tend to localize on the arch. Squamous cell carcinomas develop over the pressure points. Futhermore, when we examine the type of naevus occurring on the soles of the feet and compare it with naevi from other parts of the body, we find an important quantitative difference. Naevi on the soles, as well as those on the palms and genitalia, are almost always 'junctional'. This is important because it is the junctional type of naevus which is most liable to undergo malignant change. Why naevi on the soles and palms are usually of the junctional type is not known, but there is no reason to suspect that it is related to trauma.

Several important facts regarding the relationship of trauma to neoplasia have now emerged:

1. The long 'incubation period' which sometimes ranges over decades;

2. The fact that the tumour is often preceded by chronic and severe scarring, that is the disorganization of normal tissue relationships;

3. The wide range, and therefore the probable non-specificity, of the agents responsible for the scarring.

It is against this background that the possible role of trauma in a given case must be judged.

I would now like to turn briefly to some aspects of experimental carcinogenesis and to consider whether the results obtained in laboratory animals have any bearing on the problem of trauma and tumour formation in human beings. Associated with experimental carcinogenesis there are two phenomena which deserve special consideration.

1. *Latent Period or Induction Time.* This may be defined as the period that elapses between the first exposure to a carcinogenic agent and the appearance of the resulting tumour. It partly depends upon the intensity of the stimulus but is also related to the biological life span of each species. For example, it may be 5 months in mice, 8 months in rats and 24 months in dogs. As we have already seen, this relatively long induction time also applies to humans and it should make us extremely reluctant to accept a cause-effect relationship when a tumour develops rapidly after trauma to previously healthy tissues. Now what is supposed to be happening in the tissues during the latent period? The most widely accepted

hypothesis is that the carcinogenic agent has a direct effect on the cells (e.g. the epidermis in skin cancers) which suffer an irreversible change and become latent tumour cells. This is called 'initiation'. After a lapse of time, the initiated cells start to multiply and a visible neoplasm appears. Other workers (e.g. Prof. J. W. Orr) have put forward an alternative explanation. They suggest that the carcinogen produces more widespread changes, affecting especially the supportive and nutrient stroma. Neoplasia then develops later as a result of the altered metabolic environment of the cells. In support of this contention, Orr points out that during the latent period there is a progressive alteration of the collagen and elastic fibres of the dermis and that the rate at which this change advances is correlated with the potency of the carcinogen. Similar changes are often seen in tumour-bearing human skin. Orr has also conducted skin grafting experiments in mice painted with methylcholanthrene. Tumours were shown to develop in untreated epidermis which had been grafted on to treated sites. Conversely, grafts of treated epidermis remained free of neoplasia if they were transferred to an untreated area. It was only with thick Tiersch or whole-skin grafts that the capacity to produce tumours could be transplanted.

That an altered metabolic environment may result in tumour formation without the action of a specific carcinogenic agent is suggested by the experiments of Alexander and Horning at the Chester Beatty Research Institute. They have produced sarcomas by introducing thin plastic films subcutaneously into the abdominal wall of 6 weeks old albino rats. Tumours developed after a latent period of from 12 to 18 months and were shortest when the films of largest area were used. The tumour production is almost certainly not due to a chemical interaction between the substance of which the film is composed and the neighbouring cells. This is shown by the fact that if the same plastic material is introduced as a powder or fibres no neoplasm results. Alexander and Horning suggest that

> 'the presence of the film will create an unnatural situation in which certain cells may be subjected to a generally unfavourable environment due to lack of nutrients or oxygen, the accumulation of products of metabolism or perhaps the isolation from normal growth-controlling substances.'

Similar conditions presumably attain in the lymphoedematous limbs in which angiosarcomas develop and to which I have already referred. Experimental irradiation tumours in the skins of rats and mice are associated with chronic unstable scars and Glücksman has suggested that even here the irradiation may create environmental conditions for malignant change in regenerating cells instead of a direct malignant change in the irradiated cells and their descendants. It is therefore possible that tumours may be induced in a non-specific manner by a variety of agents which cause severe, long standing tissue disorganisation. Here the experimental

work accords with our experience in man. It is possible that trauma may be one such agent.

2. The second phenomenon to consider is *co-carcinogenesis*. By this we mean that the tumour-producing influence of a carcinogen may be augmented by that of a non-carcinogen. Thus an agent which is unable to induce tumours by itself may enhance or complete the action of a carcinogen, even if the latter is in sub-threshold doses. Such co-carcinogens include turpentine, croton oil, γ – radiation, β – radiation and heat. The tumour promoting effect of trauma was first studied by Deelman in the 1920's and the expression 'Deelman phenomenon' is sometimes used to designate this effect. It was more thoroughly studied by Linell in 1947. He showed that a single deep mechanical trauma had a distinct tumour-promoting effect on rabbits ears which had been brushed with tar for a month. Superficial, purely epithelial traumata, had no tumour promoting effect. Again we see the importance of damage to the supporting connective tissues.

Now what can we infer from this clinical and experimental evidence? Does it throw any light on the relationship of trauma to tumour formation? I would like to suggest that the following tentative conclusions are justified:

1. There is no scientific evidence that a single act of trauma to healthy tissues, followed by healing and the restitution of these tissues to normal or near normal, can cause neoplasia subsequently.

2. Trauma may act as a co-carcinogen and lead to the development of a tumour in tissues that have already been prepared by the action of a carcinogen.

3. Tumours may develop in tissues which are the seat of extensive disorganization, scarring and chronic inflammation. Trauma is one agent that can set off a train of events leading to such changes, e.g. trauma → compound fracture → chronic osteomyelitis → sinus formation → squamous cell carcinoma in or around the sinus.

In such a case, with the appropriate clinical and pathological background, the trauma may be accepted as the 'cause' of the tumour from the practical point of view. However, from a theoretical and philosophical standpoint we must bear in mind the words of Julian Huxley:

'It is surely time that we should drop mediaeval concepts concerning causation and think in terms of multiple correlation.'

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Postcontusional Syndrome

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Many of the conditions which may be found after head injury are of great psychological interest, including, e.g. delirium, Korsakow state, subdural haematoma, epilepsy, gross personality deterioration and dementia. Of greater moment, at least from the social viewpoint, is that larger group labelled (according to taste) postcontusional syndrome, postconcussional syndrome, posttraumatic neurosis, neurasthenia or psychasthenia.

In referring to this group, I shall use the term *postcontusional syndrome* as perhaps being the most widely recognized. The clinical picture is familiar and consists most characteristically of headache and dizziness

Table I: Incidence of Symptoms*

Author	*Year*	*Postcontusional Syndrome*	*Mental Changes*	*Headache*	*Dizziness*
Russell, W. R. ..	1932	—	36%	54%	43%
Lewis, A.	1942	—	70%	75%	42%
Anderson, Ch. ..	1942	—	77%	92%	86%
Symmonds, C. P. ..	1942	—	—	—	Vertigo 8%
Adler, A.	1945	—	35%	—	—
Denny Brown, D. ..	1945	15%	—	40%	34%
Ruesch, J. and Bowman, K. M.	1945	60%	50%—60%	60%	—
Guttmann, E.	1946	—	—	50%—60%	—
Wertheimer, P. and Touraine, S.	1947	—	30%	48%	36%
Mayer Gross, W. ..	1954	55%	—	—	—
Rowbotham, G. F. *et al.*	1954	—	20%	←66%→	
Muller, R. and Naumann, B.	1956	19%	—	50%	25%

* Where no figure is entered, it implies that the feature in question has not been specifically dealt with in the publication under review. Where the incidence is given for the combined symptom complex of mental changes, headache and dizziness, this will be found under *Postcontusional Syndrome* even though the author may not have used this term to designate the complex.

(sometimes with blackout) anxiety with hypochondriasis, irritability and insomnia, impaired concentration and memory. Some published figures of incidence are shown in Table I. It will be reasonable to infer that following upon head injury something between 20% and 60% of patients will at some stage show some or all of the features of postcontusional syndrome.

Aetiology

Organic Factors. It had long been pointed out that this syndrome showed, even in those of widely differing social and intellectual background, so many fundamental points of similarity, that some common organic factor must surely be involved. In an attempt to identify such a factor, many claims have been made and as many denied. Wilfred Trotter (1924) suggested that long persisting cerebral contusion might be responsible for the protracted and troublesome symptoms, and others since then have postulated the presence of diffuse punctate haemorrhages. Bielchowsky (1936) implicated a disturbed circulation of the cerebrospinal fluid and claimed also to have found evidence of ventricular dilatation in 80% of his cases. The relevance of CSF pressures has been denied by the Harvard workers (Adler, 1945; Denny Brown, 1945) but some support for the role of ventricular dilatation has been forthcoming from Davies and Falconer (1943) and Silfvverskiold (1952). Glaser reported a high incidence of vestibular involvement but Symmonds (1942) could find such evidence in only 8% of cases compared to 76% in Glaser's series. Symmonds thought that an instability of syncopal type was much more common, arising perhaps from a defect of vasomotor control resulting from medullary concussion. Penfield and Norcross (1936) attributed headache to the formation of adhesions in the subdural space. Williams (1941), while emphasizing the limitations of the technique, believed that the high incidence of EEG abnormality in these cases was an indication of organic cerebral abnormality. Ruesch and Bowman (1945) suggested that a possible continuing disturbance of the function of the adrenosympathetic system might be involved or that the brain injury might affect the nervous and hormonal control of extra cerebral organs such as the thyroid and thus account for some of the symptoms. McConnell (1956) attributed the clinical picture to subdural hygroma and claimed to relieve it by burrhole evacuation. Sachs (1957) has recently drawn attention to the role of acetylcholine and serotonin in the acute stage of head injury—a matter of some interest perhaps in the present connection in view of the role of serotonin in mental disorder.

Psychogenic Factors. The psychogenic factors have been stressed among others by:

Russell Brain (1942): '. . . a severe head injury is not an important cause of traumatic neurosis';

Jefferson (1944): '. . . the post-traumatic syndrome is always neurotic when no blood is found in the CSF';

Ruesch and Bowman (1945): '. . . the importance of organic brain damage is overrated . . . after one year most of them are purely personality problems'; and

Muller and Naumann (1956): '. . . the post-traumatic syndrome is predominantly a psychogenic disorder.'

Those who lean towards a psychogenic interpretation of the condition draw attention to

i. The apparent lack of correlation between the severity of the injury as judged by the posttraumatic amnesia, and the occurrence of posttraumatic syndrome;

ii. The frequent predisposition to neurosis to be found in the previous history;

iii. The clinical similarity to other neurotic disturbance without brain damage;

iv. The apparent motivation in so many cases; and

v. The improved prognosis with psychologically orientated methods of rehabilitation.

Taking these points in order, we find first a considerable measure of agreement about the lack of correlation between severity of injury and development of symptoms (Adler, 1945, Denny Brown, 1945; Russell, 1954.) It is clear that the duration of the posttraumatic amnesia as a prognostic indicator of disability failed as far as the chronic posttraumatic syndrome was concerned. Guttmann (1946) and Muller and Naumann (1956) found that headache was not related to the severity of the injury. Walker (1958) dissents from the view that symptoms of the kind in question do not follow severe injury. Jefferson (1944) and Ruesch and Bowman (1945) contrast the progress of symptoms in head injury with those following controlled surgical assault on the brain where the symptoms of headache and dizziness are at first maximal and then gradually recede. The importance of neurotic predisposition has been stressed by Symmonds (1942), Symmonds and Russell (1943), Ruesch, Harris and Bowman (1945), Muller and Naumann (1956) and Grunstad (1958). Lewis (1942) concluded that the longstanding and relatively intractable postcontusional syndrome was apt to occur in much the same sort of person as develops a psychiatric syndrome in other circumstances without any brain injury at all. Denny Brown (1945), on the other hand, found little correlation between pretraumatic psychiatric abnormality and the occurrence of posttraumatic 'mental' symptoms, although he adds that headache and dizziness, as such, were associated with pretraumatic nervousness; and Adler (1945) found as many of normal personality developing symptoms as those of abnormal personality. This was also McConnell's (1956) experience, although he and they would acknowledge that once the symptoms have developed, the pretraumatic personality will to a considerable extent determine the course. The clinical similarity between the postcontusional syndrome and other neuroses without brain damage has been pointed out by Ruesch and Bowman (1945), Lewis (1942), Denny Brown (1945) and others. As to the significance of environmental factors, of which more will be said later, it has generally been considered to do with occupational, domestic and financial difficulties, and to have been, from the prognostic point of view, a better indicator than the severity of the injury (Guttmann, 1943; Adler, 1945; Denny Brown, 1945; Ruesch and Bowman, 1945; Muller and Naumann (1956). Speaking of rehabilitation, Jefferson (1944)

thought that the key to the situation lay in the close association of neurosurgeon and psychiatrist and the importance of a recognition of psychological factors is widely accepted.

A Total Reaction. Are we then to take up a stand on the postcontusional syndrome being either organic or psychogenic. Certainly attempts have been made, on clinical grounds, e.g. by Schaller (1939) and by Schilder (1949) to segregate two posttraumatic syndromes, the one organic and the other psychogenic. Lynn (1945) claims to have devised a psychological test battery capable of making this distinction in clinically difficult cases. Lewis (1942), commenting upon earlier attempts to devise such a battery, had observed that where the diagnosis of organic damage was clinically doubtful, no psychological test could be relied upon to supply an unequivocal answer. Thus Chapman (1957) has recently shown that two groups of patients, the one subjected to prolonged emotional stress, the other to known cerebral tissue loss, have demonstrated, on refined tests of intellectual function, the same kind and degree of intellectual deficit.

In the postcontusional syndrome, as it generally presents, we should not expect to find other than very fine impairment and it would seem that tests will not help in such a case; nor either in the usual case will we expect to find confirmatory evidence of organic damage in the shape of gross neurological signs. One cannot, on such a basis, then conclude that there is not or has not been some organic dysfunction underlying the development of the symptoms. Nor is it possible, on the basis of the individual symptoms, e.g. headache, as seen in the postcontusional syndrome, to distinguish between the organic and psychogenic varieties (Anderson, 1942; Symmonds, 1942).

It is misleading and certainly therapeutically sterile to attempt a clear dichotomy into organic and psychogenic types of the postcontusional syndrome. The individual's personality, intimately associated as it is with cerebral function, cannot be divorced from mild impairment of that function. The clinical picture in cerebral dysfunction, be this fine or moderately severe, does not represent the simple arithmetical sum of intact and damaged faculties. For besides any symptoms which are the direct expression of local cerebral abnormality, there will be others of a more indirect kind which express the attempt of the personality as a whole to cope with this deficiency and to avoid the disturbances of the catastrophic reaction (Goldstein, 1942).

The clinical picture therefore will depend firstly on the personality and secondly on the degree of cerebral disturbance, and it will be further modified, as we shall see, by environmental factors. It is not, therefore, a question of either organic or psychogenic but rather what proportion of each. The more serious is the organic disturbance, the less will be attributed to psychogenic aetiology. The less serious the apparent organic disturbance, the more will we search for light in the previous personality

and the personal environment. Few cases indeed will be found towards the extremes of the continuum where it is possible to assert that either organic factors or psychogenic factors are entirely responsible. We must remember that the interplay of organic and psychogenic factors is the rule and not the exception in cerebral disturbance of the milder kind. Thus Wolff and Curran (1935) have demonstrated that in delirium, the clinical picture is strikingly affected by the previous personality but, as might be expected, the individual differences become blurred as the disorder increases in severity. Observations on temporary and isolated deviant sexual behaviour in a group of confused neurosurgical patients have shown that this behaviour was highly correlated with previous sexual maladjustment (Weinstein, 1954). It is also well known that to explain the discrepancies between the pathology and the clinical picture in individual cases of senile dementia and cerebral arteriosclerosis, we must have regard to personality and social factors (Rothschild, 1937; Williams, 1942; Post, 1944). Sands and Rothschild (1952) saw the clinical picture in these cases as ' the product of special vulnerabilities in the personality coupled with sociopsychiatric stress in the presence of organic cerebral disease '—a formulation I submit which goes very near to describing the average case of postcontusional syndrome.

We will not therefore conclude in the individual case that because personality factors or social stresses appear to be operative, that there is no organic component. In spite of its intrinsic difficulties we may, both for therapeutic and medicolegal purposes, have to attempt in the case being examined, to assess the relative contributions of psychogenic and organic factors. Here we can only be approximate in our estimation and as Walker (1955) has put it:

'. . . I fear that at present the opinion as to the dominance of functional or organic factors, in the absence of positive signs, is based upon the unanalysed impression made by the patient upon the examiner.'

However, as a rough guide, we may believe that the less severe the accident as judged by the posttraumatic amnesia, the less stable the previous personality, the more exaggerated or inconsistent the symptoms, the more responsive the patient's improvement to changes of surroundings or circumstances, the more obvious the contemporary social stresses which might account for the continuation of the symptoms, the greater the secondary gain—the more likely is the illness one to which psychogenic factors make the greatest contribution. The converse is also true and the presence of demonstrable intellectual impairment with intolerance to heat and alcohol is confirmatory evidence of a significant organic contribution. But these methods cannot be more than crude.

Apart from its contribution to the posttraumatic picture, the patient's personality may, in the socalled ' accident prone ' individual have actually conduced to the accident. In Ruesch and Bowman's series (1945) some 25% of the cases had had previous accidents. Repeated involvement in

accidents is thought to represent some abnormality of personality and accident prone people have been described as being often of resentful, impulsive and hostile disposition. Some may be impelled by unconscious feelings of guilt to seek selfpunishment and others to seek destruction in an attempt to escape from some intolerable dilemma. It will be appreciated that, in such cases, the causes which led to the accident may very well prolong the disorder. Naturally, the elderly, the feeble minded and the alcoholic, as well as those who are either morbidly preoccupied or excited, are more prone to accidents than are others.

Something further needs to be said on environmental factors. It may be that the accident has quite fortuitously resolved certain social or economic difficulties. Thus the patient may, until the time of the accident, have been working under stress either because he was not equal to his job or because he resented the conditions of his work. He may then be freed, in an unconscious way, from the obligation to continue and the symptoms are thus hysterically utilized—although not necessarily produced in this way in the first instance. It should be remembered that what appears to be grossly hysterical behaviour may be an elaboration and exaggeration of existing physiogenic defects or at least it may have been precipitated by such in the first instance.

Compensation as a Factor. Much has been said of compensation as a factor and no doubt it is of some significance. Unfortunately too many people speak, in the same breath, of malingering. Malingering, in the sense of conscious deceit for gain, is thought to be very rare in head injury —estimated as a rule at less than 1% (Wechsler, 1935; Anderson, 1942; Goldstein, 1942; Mayer Gross, 1954). Most workers assert that the role of compensation, at least on a conscious level, has been greatly overrated as a factor in the development of the picture, although it may arise later when prolonged disability has brought its own financial problems (Lewis, 1942; Ruesch and Bowman, 1945; Mayer Gross, 1954; Muller and Naumann, 1956). Much has been made of the relatively greater incidence of postcontusional symptoms following accidents at work than on the road. Russell Brain (1942) pointed out that an injury sustained at work suggested that the man's job was dangerous and was therefore a special threat to his security and further that the man felt constantly anxious about his economic position since the provisions of the Workman's Compensation Act meant that his disability award was constantly liable to dispute. Russell (1934) reported that 83% of his noncompensation group were back at work in 6 months while 35% of the compensation group were still idle after 18 months. This, of course, may merely mean that many of the others were forced because of their economic position to return too soon (as we shall see, a follow-up a couple of years later revealed that they were not necessarily well—more than 50% had symptoms 18 months later). There is further a widely held belief that compensation is all they want and that

they will recover promptly on receiving it. There is a most curious lack of evidence on this point and therefore not much reason to suppose that it is true. Compensation may have a purely symbolic value for the patient in so far as it represents the employer's interest and sympathy and desire to help. Conversely, in resentful patients who feel that they have had a raw deal, it may be associated with a desire to punish the employer.

Finally there will remain, no matter what the individual circumstances, certain fairly common psychological reactions to head injury as such. The head is perhaps the most important part of the body image and most people, if asked, would there locate their personality, their 'real self'. Injury to the head then is a special insult to their psychic integrity and their sense of security. The development of such symptoms as dizziness, poor concentration and impaired memory aggravate the sense of insecurity. Unconsciousness (with its relation to the concept of death) and confusion, enhanced by the sometimes bleak and aweful surroundings of a neuro-surgical ward, all combine to impress upon the patient the supposedly grave nature of his condition and the threat to his life. Small wonder then that an introspective scrutiny develops, the patient fearing, perhaps, that a blood clot is forming on his brain, or that he may become paralysed or insane or that his intellect may be permanently damaged. Many who entertain such fears are reluctant even to voice them as if to do so were to make the spectre more real.

Prognosis and Rehabilitation in Head Injury

The principles of rehabilitation have been outlined by Jefferson (1942), Lewis (1942), Brain (1942), Cairns (1943), Muller and Naumann (1956) and others. It will involve an appreciation of the man's psychological as well as physical needs. Early physical activation is favoured together with a definite assessment of the man's disabilities so that retraining may be planned where necessary and a firm prognosis given for disability. From the psychological point of view the emphasis will be on the assessment of personality and social stresses, and no doubt, more may be done in this latter area than elsewhere. Deep psychotherapy is unlikely to be involved. A supportive approach will include suggestion, reassurance and persuasion. Group therapy may be of considerable value also. The patient may be most amenable to psychotherapy on first regaining lucidity when he will be most suggestible.

The true prognosis in head injury, as far as actual clinical recovery from symptoms is concerned, is difficult to estimate, since most of the follow-up studies rely, not upon such recovery, but upon a period of disablement from work—this at least is a measureable feature of value both to the individual and the community. From a perusal of the published work, it seems that periods of disability vary within fairly wide limits in different

material. Something between 3% and 20% may not return to normal employment following head injury; of those who do return the majority will go back within the first 4 weeks. The average loss of working time for those who eventually return to work has been given in different series as between 8 and 18 weeks. It appears to be generally agreed that prolonged disability following head injury relates to the group of symptoms which we have been discussing under the postcontusional syndrome (Guttmann, 1943; Adler, 1945; Denny Brown, 1945; Wertheimer and Touraine, 1947; Russell, 1954; Rowbotham, 1954; Muller and Naumann, 1956). Return to work is, however, not necessarily synonymous with clinical recovery and may merely indicate a willingness on the part of the patient to tolerate some headache, dizziness or memory impairment. (The latter would clearly be of much greater moment to an intellectual worker). Studies of symptomatic recovery as such are therefore of great interest but are relatively fewer in number. Reported figure for the persistence of symptoms after discharge are given by Russell (1934) as 55% after 6 months and 40% after 18 months; by Cedermark as 24% (1942) after 3 years; by Guttmann (1946) as 20% after 6 months; and the most encouraging by Naumann and Muller (1956) as 8% after one year. Anderson (1942) thought that the tendency was towards recovery in from 2 months to 2 years. Such findings will perhaps temper the facile optimism engendered by studies which are addressed only to period of disablement.

Summary

In its frequency of occurrence and its social and economic repercussions, the postcontusional syndrome is perhaps the most serious of the possible sequelae of head injury. An interpretation which seeks to explain this clinical phenomenon on either a purely psychogenic or purely organic basis is likely to do violence to the facts and is certainly conducive to therapeutic sterility. In each case it will be a question of assessing the relative contributions made by organic and psychogenic factors—the assessment will in any event be crude. At the extremes of the 'psychogenic-organic continuum' we shall find perhaps 1% of malingerers to the left and some 2% to 3% of gross organic intellectual or personality deterioration to the right. The organic factors involved may not necessarily be the same in all cases, nor either will the psychogenic factors..

It is emphasized that the role of compensation as an aetiological factor has been exaggerated—there is very little evidence to show that those who are paid compensation are thereby relieved of their symptoms.

Studies of clinical recovery as such suggest that the symptoms in this condition are more chronic than might be inferred from those studies which deal only with periods of disability from work.

The conception of the 'whole man', important in the treatment of any human ailment, is perhaps nowhere more vital than in the field of head injury, for the human personality intimately associated as it is with cerebral function can hardly be divorced from an alteration of that function.

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The Association of Trauma and Spondylosis

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Spondylosis literally means 'a state of having vertebrae;' but it is a well-defined clinical entity due to acquired deformity of the spinal structure. This deformity may be symptomless or may give rise to pain or paralysis (due to involvement of the spinal cord or nerve roots) varying according to the anatomical site of the lesion.

Pathologically, degenerative changes occur in two types of joint: the synovial diarthrodial joints and the intervertebral amphiarthrodial joints.

Histologically, degenerate annulus is present between the osteophytes at the margins of the vertebral bodies.

Spondylosis is caused by fair wear and tear in the natural ageing process or earlier by unfair wear and tear due to congenital or acquired conditions affecting the spinal mechanism.

Examples of the effect of trauma on established cases of spondylosis were shown to illustrate the various ways in which the spinal cord or nerve roots could be involved. Extension injuries usually caused the greatest damage. A rubbery protrusion might cause a cord contusion, whereas a firm protrusion might produce a myelopathy by embarrassing the blood flow through the anterior spinal artery.

It was emphasized that the Forensic Pathologist could assist the clinician in furthering knowledge of the condition.

Accumulation of Enzymes and Organic Acids in Cadaver Blood

Dr. J. B. Enticknap, East Ham Hospital

Dr. Enticknap's paper had already been accepted for publication by the *Journal of Forensic Medicine** prior to the meeting, but he added:

Fatty acids accumulate in cadaver serum in a closely analagous fashion but the slope of the rise is very much less sharp. The increase was about 1 m/E/1 per 12 hours. Further, the mean values of the serum fatty acid content in the different disease groups held considerable promise of providing a biochemical means of distinguishing some of the different groups of fatal heart attacks.

* Dr. Enticknap's paper was published in this *Journal*, Vol. 7, No. 3, 1960, pp. 135-146.

Self-Strangulation

Dr. C. Keith Simpson
Guy's Hospital, London

Dr. Keith Simpson spoke of 4 cases of self-strangling, to be published in another journal. Following Dr. Simpson, the following members quoted similar cases: Dr. F. Camps, Prof. C. Polson, Dr. E. Rentoul, Dr. R. D. Teare, Dr. Hocking.

Carbon Monoxide Poisoning

An Unusual Case

A. Keith Mant, M.D.

Department of Forensic Medicine, Guy's Hospital, London, S.E.1.

The dangers of combustion in inadequately ventilated spaces have been known from remote times, and the deaths in Great Britain from this cause recall fatalities described in some of the earliest medical writings. Whilst it is probably true to say that all those used to dealing with sudden deaths —especially police surgeons and coroners' officers—are fully aware of the dangers of incomplete combustion, it is equally true that few appreciate how little combustible material need be burnt in a vitiated atmosphere, or under conditions of inadequate ventilation, to produce a fatal concentration of carbon monoxide. The case I am about to describe fully illustrates this point.

Mr. H., a night-shift worker, returned home one morning in February of this year, to find his wife, aged 39 yrs. who had been perfectly fit except for a slight cold when he had left her the evening before, lying dead on the kitchen floor. She had apparently fallen forwards off the chair from which she had been watching television. The television set was still turned on. Three dogs, all of above medium size, also lay dead in the room.

1

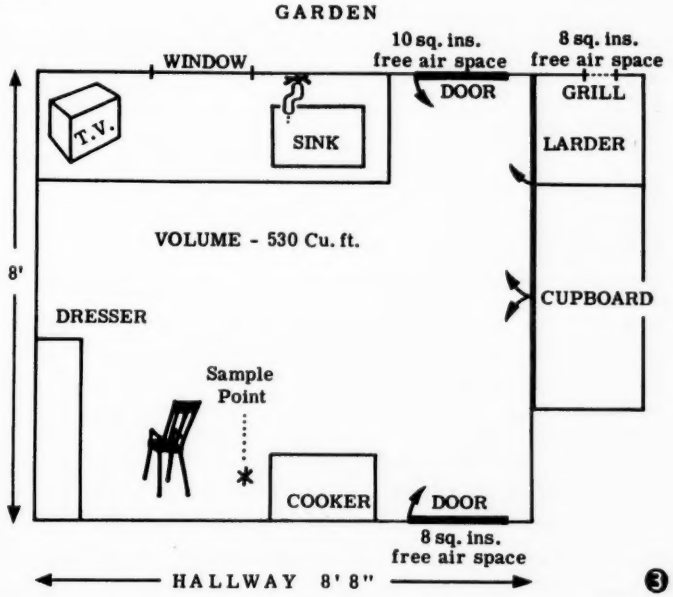
2

Mr. H. noticed a sweet burning smell but no smell of gas. On the gas stove, over a partly turned on and lighted burner, was a bucket which had burnt dry (Figs. 1 and 2), and the clothing inside, which consisted of six handkerchiefs, was charred. He immediately turned off the gas and called an ambulance. The police sergeant, who arrived quickly upon the scene, noticed a sickly odour in the kitchen but no smell of gas.

The body was taken to the public mortuary, and within two hours of the discovery of the fatality an autopsy was carried out.

The body was well nourished and of a bright pink colour. There were no injuries beyond a superficial pressure abrasion on the chin, reproducing the weave of a coconut mat which was beneath the deceased when she was found. There was no natural disease present. An examination of the blood with a Hartridge reversion spectroscope showed a 73% saturation with carboxy haemoglobin. The blood from one of the dogs was also examined, and this had a concentration of 71% carboxy haemoglobin.

Meanwhile the gas installation and apparatus had been examined by the South Eastern Gas Board and had been found to be sound and efficient in all respects.



The kitchen (Fig. 3) was on the ground-floor and measured approximately 8′ × 8′ 8″ × 8′, having therefore a cubic capacity of 530 cu. ft. There was a tightly fitting casement window and two doors to the room. Gaps between the outer door and the frame amounted to some 10 sq. in., and between the inner door and the floor to some 8 sq. in., though this latter gap could have been almost obliterated by the mat. There was no air brick in the kitchen, but a small grill in the larder wall had a free area of 8 sq. in.

The bucket was of zinc and was of the standard domestic pattern. It had a flange around the base, some $\frac{7}{8}$″ deep, which could be expected to interfere with the complete combustion of coal gas at least whilst the bucket contained cold water and the gas was fully turned up.

A number of tests were carried out to ascertain the concentration of carbon monoxide which might be built up in the air under conditions similar to those pertaining at the time of the fatality. Observers were in the kitchen for relatively short and long periods during these tests, and the physiological effects, if any, were recorded. It was considered, however, that at the times these tests were carried out the air movement in the kitchen was greater than on the night of the accident, not only because of different external climatic conditions, but also because observers had to enter and leave the room in order to take samples of air.

Table 1 (Test A): 4 Pints of Water (Depth 2¼") Boiled Dry on Stove

Time Hr. Min.	*Burner*	*Gas Rate Cu.ft./M.*	*Water*	*% CO—at Sample Point*	*Water Depth*
0	Full On	22·5	52°F	0·00	2¼"
15	Full On	22·5	Boiling Commenced	0·06	—
21	Simmer Rate	7	Simmering	—	—
30	Simmer Rate	7	Simmering	0·03	1½"
2—00	Simmer Rate	7	Dry	—	Nil
2—34	Simmer Rate	7	Dry	0·025	Nil

In the first test a half-gallon water was boiled dry over a large burner, and samples of air were taken at intervals from a point 6 inches from the stove. The gas burner was turned up fully until boiling commenced and then reduced to a simmer. It will be seen from Table 1 that the maximum concentration of 0.06% carbon monoxide in the atmosphere was reached by the time the water commenced to boil. After this the gas burner was turned to simmer and the carbon monoxide content fell to 0.03%. When the water had evaporated it fell still further to 0.025%.

An observer was in the room during part of this test and felt no ill effects.

In the second test two observers stayed in the closed kitchen whilst half a gallon of water was brought to the boil and simmered for nearly one hour. One observer sat in the chair close to the stove. Neither observer experienced any physical distress other than some discomfort due to the heat and high humidity.

The third test was carried out in the laboratory, and varying numbers of handkerchiefs were slowly charred at the bottom of the bucket over a burner turned to simmer, consuming 7 cu. ft. gas per hour. Charring commenced after one minute with the evolution of carbon monoxide. As the charring progressed smoke became denser, and carbon monoxide was evolved more rapidly. Tests showed that carbon monoxide was also produced when the washing powder used by the deceased was charred.

The fourth test (Table 2) was carried out in the deceased's kitchen. A quantity of fabric equivalent to six medium-sized handkerchiefs was saturated with water and spread over the bottom of the bucket. The bucket was then heated with the burner turned fully on for three minutes, after which it was turned to simmer. Observations were made, and samples of air taken as shown in Table 2. It will be seen that after half an hour a fatal concentration of carbon monoxide was built up in the kitchen, and this persisted for at least half an hour. One of the observers who entered the kitchen for a short time in order to obtain a sample of air suffered a severe headache and malaise.

Table 2 (Test B): Incineration of 6 Medium sized Wet Handkerchiefs

Time Hr. Min.	*Burner*	*Gas Rate Cu. ft./M.*	*% CO—at Sample Point*	*Observations*
0	Full On	22	0·0	
3	Simmer	7	—	Early Charring
15	Simmer	7	0·06	Little Smoke
30	Simmer	7	0·09	Dense Smoke
40	Simmer	7	0·12	Smouldering
55	Simmer	7	0·10	Smouldering
85	Simmer	7	0·06	

When the deceased was found, five cigarette stubs were present in the ash tray on the dresser by her side, and the smoking of these cigarettes could have been an additional contributory factor to the death.

If the bucket had been covered by a lid the combustion of the clothing would have been even less complete, and a very much higher concentration of carbon monoxide would have been reached in the atmosphere.

One surprising feature of this case is the fact that neither the deceased nor the dogs were apparently roused into activity by the smoke produced. A possible explanation is that the gas was flowing at a much lower rate than was used during the investigations. Under these circumstances there would be less smoke, and more carbon monoxide evolved.

I should like to thank Mr. J. B. Carne, Head of the Appliance Laboratory of the South Eastern Gas Board, for the details of the investigations undertaken by his Laboratory.

Discussion

Dr. G. Forbes felt that we were all alive to the dangers of these paraffin stoves in confined spaces, and that it was a particularly useful point to observe in the case of many Coloured people who come to this country feeling the cold tremendously, getting into little bedrooms, turning on these heaters and then going to sleep.

Recently he had a case of a Coloured man lying dead in bed in a small room which he had rented. He had not been seen for a couple of days. The case was treated at first as an ordinary sudden death, but then numerous enquiries were started regarding a heater, etc. and a paraffin cooker was found in the room. However, no carbon monoxide was found in the blood and it was finally established that he had died from sickle cell anaemia.

Death from Massive Ischaemic Necrosis of Muscle

D. P. Winstanley
St. George's Hospital, London, S.W.1.

The lesson of this case is that the surgeon, in attempting to save a limb, may lose a life.

Case Report

A butcher's boy of 19 years was cutting the bone out of a joint of meat with a pointed knife 6 inches (15 cm.) long when it slipped and entered his right groin. When he arrived at the hospital, there was brisk arterial bleeding from a small stab wound 1½ inches (4 cm.) below the inguinal ligament. It was controlled by digital pressure, with some difficulty, and as operation could not be begun until nearly 7 hours after the accident, digital pressure on the artery had to be kept up for the whole afternoon. A blood transfusion was set up and the blood pressure remained at 105/60 mm. Hg.

At operation the femoral artery was found to be almost completely severed just above the profunda; the femoral vein had also been penetrated. Both were sutured, circulation being restored about 8 hours after the injury; 8 pints of blood were given in all.

Next day his general condition was fair but he passed no urine. On the second day 22 oz. (620 ml.) of urine were obtained by catheter. The specific gravity was 1014 and it contained a trace of protein, but no myoglobin. The blood urea was already 100 mg. per 100 ml.

On the third day his condition deteriorated. He passed only 1½ oz. (40 ml.) of urine. The blood urea was 116 mg. per 100 ml.; no record of the blood electrolytes is available. His respiration became irregular and gasping and he died at 9 p.m., 79 hours after the injury.

Post-Mortem Findings

The stab wound in the right groin was quite small, not more than ⅜ inch (1 cm.) long. The femoral artery was narrowed at the suture, but the lumen was obviously wide enough to maintain the circulation. The most striking change was in the muscles of the thigh and calf; they were all pale yellowish brown in colour, in striking contrast to the healthy muscles of the other side.

The renal cortex was pale and swollen and the lungs acutely oedematous.

Histological Findings

Thigh Muscle. The muscle fibres from the affected right thigh had lost all their nuclei, but transverse striation was quite well preserved. There were a few sharply defined segments where the cytoplasm was replaced by grey spongy material. Between the fibres there were many polymorphonuclear leucocytes, some of which were breaking down. In some areas they formed dense clumps. The changes resembled those of early myocardial infarction.

Kidney. An unfortunate delay of 3 days between death and examination led to such severe autolysis of the convoluted tubules as to obscure any ante-mortem necrosis. There were, nevertheless, many casts in the distal convoluted and collecting tubules. They had a brownish tinge; some were granular and others homogeneous Perl's and Turnbull's stains failed to show any free iron in them.

Discussion

This man is not the first butcher to injure himself in such a way. In a precisely similar case,[1] a through-and-through puncture wound of the common femoral artery was dealt with by end-to-end anastomosis and the limb was saved. The interval between injury and operation was only 4½ hours. In the present case, however, the 8-hour period of ischaemia was evidently long enough to cause massive necrosis of the muscles of the lower limb. By the time the artery was sutured the muscles were irretrievably damaged and the restored blood flow served merely to wash into the circulation the products of muscle breakdown, among the most important of which is potassium.

Renal failure was not the immediate cause of death. When he died the blood urea was still comparatively low, and it is well known that cases of total anuria may survive for 2–3 weeks, the blood urea rising over 400 mg. per 100 ml.

Death at this early stage was presumably due to massive release of potassium from the necrotic muscle. This affected the myocardium, finally causing cardiac arrest. The renal failure contributed in that no excretion of potassium was possible.

Muscle ischaemia of the same kind can be produced by prolonged application of a tourniquet. In an unpublished case[2] a tourniquiet was inadvertently left on the thigh for several hours after a meniscectomy. The patient, a man of 31, developed anuria and died 3 weeks later. Tourniquet experiments in animals have produced temporary anuria, but this has never lasted long enough to cause death; possibly the tourniquets have not been left on long enough—2–3 hours is the longest period described.[3] It seems likely

that the human kidney is more sensitive to injury by functional ischaemia than is the kidney of the dog.[4]

In this case the surgeon was obviously in a grave dilemma. If the delay in getting the patient to the operating theatre could have been foreseen at the outset, it might have been better to ligate the artery and vein. This would have saved the man's life, but he would probably have lost the limb.

In the post-operative management of such cases a watch must be kept for myocardial changes due to potassium release. Daily electrocardiograms and electrolyte estimations are essential. It may be necessary to undertake dialysis at an early stage, even when the blood urea is still comparatively low, to get rid of an excess of potassium.

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The Detection of Diatoms in the Bone Marrow as Evidence of Death by Drowning

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It is now generally accepted that, during drowning in water containing microscopic plankton (diatoms, algae, plant fibres, etc.) the latter penetrate in small quantity into the lung capillaries, reach the left heart and are thus dispersed throughout the body by the arterial circulation. It is not our intention to dwell here on the research, extending over about half a century, which proved conclusively the reality of this penetration. We refer the reader to our paper entitled *Gerechtelijk-geneeskundige Problemen bij de Verdrinkingsdood* (Verhandelingen van de Koninklijke Vlaamse Academie voor Geneeskunde van België, 1960, **XXII,** 9–23), as also to our article on drowning in the *Ciba Symposium,* Vol. 9, No. 4, 1961, in both of which he will find a cross-section of the basic historical data, with the bibliographical sources.

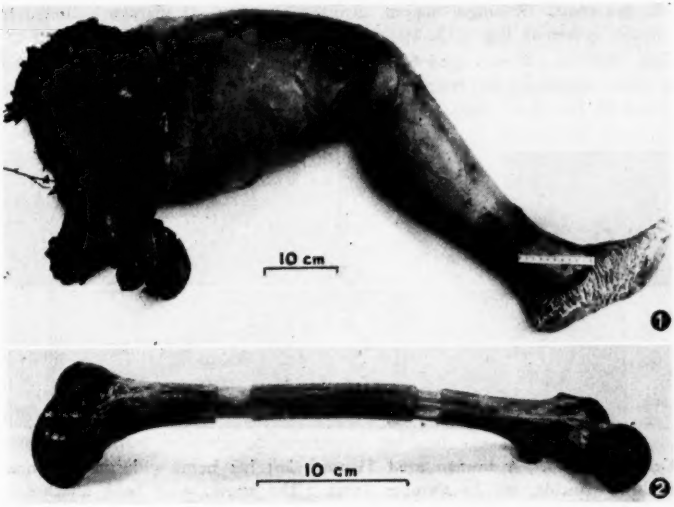
At the Second International Meeting on Forensic Pathology and Medicine, held in New York on 18 September 1960, we presented a discussion of the technical problems involved and described the methods which, in our hands, have given the most satisfactory results for the detection, in the heart and in the lungs, of microscopic plankton and of diatoms specifically in the brain and, eventually, in other organs. (A summary of this paper is available in the Abstracts of the Meeting.)

To the Hungarian forensic pathologist L. Tamaska goes the undoubted credit of having, as far back as 1947, provided a new approach to the diagnosis of death by drowning by demonstrating the possibility of detecting diatoms in the human bone marrow. Thanks to his method, it is sometimes possible to reach a conclusion in cases where the examination of the lungs and the heart has become useless because the cavities of the body have collapsed following advanced putrefaction or have been ripped open, and even when only a limb is available (as in the case illustrated in Fig. 1).

We have not been able to read the author's original paper, in Hungarian, which is referred to briefly in an article by J. Incze, L. Tamaska and J. Gyöngyösi, published in 1955 in the Dtsch. Z. ges. gerichtl. Med., **43,** 517. As the waters of the rivers of Hungary appear to be exceptionally rich in diatoms, we have felt it advisable to repeat these investigations in Belgium, where this is usually not the case, both the coastal seawater and that of the rivers containing at the utmost only moderate amounts of plankton, probably because of the high degree of chemical pollution. We are not acquainted with Tamaska's technique, for the reason given. We therefore

worked out our own. It was demonstrated by means of a film at the New York meeting.

We have, since then, pursued our research, of which we present a survey to date in this paper. Our investigations were limited to the marrow of the femur, the tibia and the humerus, the collection of which can be achieved without risk of contamination from the outside by diatoms present on the skin and clothing of the body.



Our technique can be summed up as follows. One or more long bones are extracted from the body. All soft tissues attached are cut off and the bone scrubbed thoroughly. Halfway along the shaft, for a distance of a couple of centimetres, a layer of bone is cut out to a depth of about 2 mm., by means of a machine tool. Alternatively one can also perform this operation in 2 different places nearer the epiphyses (Fig. 2). This procedure effectively prevents contamination of the bone-marrow. The shaft is then sawn through at the sites marked out by the machine tool. The bone marrow can now be collected by means of a gynaecological curette of adequate size. Experience teaches that from 15 g. to 40 g. of marrow can thus be recovered. The marrow is placed in a Kjeldahl flask in which it is chemically digested by adding small quantities of nitric acid at a time. Sulphuric acid is contra-indicated because it produces precipitates which completely obscure the microscopic picture. Heating is best done with an ordinary Bunsen burner. The operation lasts 1–2 hours and yields a transparent yellow fluid with a supernatant disc of fat. The yellow fluid is next centrifuged. The deposit (usually hardly visible to the naked eye) is poured on to a slide and examined, while still wet, under a coverglass. The examination of the fat disc, after prior solution in ether, has never rewarded us for our pains.

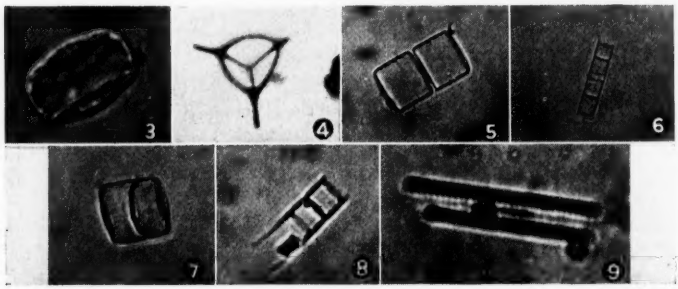
The result of the investigation depends entirely on the quantity of diatoms present in the water. It is therefore wise to examine the latter

beforehand. If the water is poor in diatoms, it is hardly worth persevering in one's endeavours.

The following series of cases illustrates our own results. We only present the data provided by the investigation of the long bones, it being understood that, in all our cases, microscopic plankton was searched for routinely in the lungs and in the heart (except, of course, in Case 1960/245) and diatoms in the brain.

Case 1960/37. A woman aged 32 years committed suicide by drowning on the North Sea coast. A single diatom, detected in 15 g. of marrow collected from the femur, is seen in Fig. 3 (X 1600; *size of diatom*: 12 μ x 9 μ).

Case 1960/83. A man aged 62 years committed suicide by drowning in a pond. One single silicoflagellate, recovered from 20 g. marrow collected from the femur, is shown in Fig. 4 (X 440).



Case 1960/245. A woman aged 51 years left her home with the intention of committing suicide, on 23 October 1960. The trunk, with head attached, was recovered from a nearby river 5 days later. The chest having been ripped open by a propeller, proof of death by drowning was sought and found by the examination of the brain, for diatoms. On 5 November 1960 the complete left lower limb was recovered (Fig. 1); 15 g. of marrow were collected from the femur. They contained numerous Melosira of various sizes, two of which are shown in Figs. 5 and 6 (X 520).

Case 1960/252. The body of a man aged 49 years was recovered from the river Scheldt 7 days after his disappearance; 25 g. of marrow, collected from the femur, yielded numerous diatoms. Two Melosira are visible in Figs. 7 and 8 (X 900).

Case 1961/41. The body of a man aged 49 years was recovered from the river Scheldt 21 days after his disappearance; 30 g. of marrow, collected from the femur, yielded one diatom (a complete Synedra frustule), shown in Fig. 9 (X 1200).

We were given the opportunity of examining the long bones of the body of a man who had not drowned, but had been buried at sea (Case 1959/214). It stranded on the beach, east of Dunkirk, after a heavy storm. Notwithstanding an exhaustive investigation, the marrow did not yield *one single diatom*. This negative result emphasizes the full significance of a positive finding.

From the data presented it is obvious that the method described by Tamaska can be expected, be it but in a restricted number of cases, to render invaluable service to the forensic expert.